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Review

The amygdala complex: Multiple roles in associative learning and attention

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ABSTRACT **Although certain neurophysiological functions of the amygdala complex in learning seem well established, the purpose of this review is to propose that an additional conceptualization of amygdala function is now needed. The research we review provides evidence that a subsystem within the amygdala provides a coordinated regulation of attentional processes. An important aspect of this additional neuropsychology of the amygdala is that it may aid in understanding the importance of connections between the amygdala and other neural systems in information processing.**

Kluver and Bucy (1) observed that monkeys became remarkably tame after surgical removal of the temporal lobes, a phenomenon noted as early as 1888 by Brown and Schaefer (2). Subsequent research extended these observations to indicate that the amygdala complex plays an important role in emotional behavior. This conceptualization of amygdala function led to subsequent research on the role of the amygdala in emotional learning (3).

The Role of the Amygdala Central Nucleus (CN) in Associative Learning

Dating from Kluver and Bucy's (1) classic studies, many lines of research have come to support the tenet that neural processing within the amygdala complex is important for assigning emotional significance or value to events through associative learning. For example, in animals with amygdala damage, a cue that signals an impending aversive event (such as footshock) fails to elicit fearful behavior (4, 5). Learned associations between cues and positive experiences are also deficient in animals with amygdala damage. In one commonly employed task, rats normally learn to seek, or remain in, an environment associated with reward, a phenomenon referred to as conditioned place preference. Rats with amygdala damage fail to show this form of associative learning (6). In the past decade, the study of acquired emotional responses has yielded much information about the neural circuits in the amygdala that are involved in associative learning.

In addition to a well-established role in emotional learning, specific circuitry within the amygdala complex contributes to the regulation of attention. The research that led to this view of amygdala function began with an exploration of the role of the central nucleus (CN) of the amygdala in Pavlovian appetitive conditioning (7). Removal of CN neurons was produced by microinjection of the neurotoxin ibotenic acid, which spared fibers of passage and neurons in adjacent amygdala nuclei. In our behavioral studies, rats received training in which presentations of an originally neutral cue, such as a tone or light, were followed by delivery of food at a recessed cup located in a standard animal test chamber. Rats with CN lesions learned as readily as intact rats; in the presence of the cue, rats approached the recessed cup in anticipation of food delivery. Our result suggests that this nucleus of the amygdala is not necessary for learning the motivational significance of a cue signaling food reward. If this is the case, then an important functional distinction between subsystems within the amygdala complex might be possible. In contrast to our results, McDonald and White (6) found that damage to the lateral nucleus of the amygdala abolished the acquisition of learned cue preferences in rats. The basolateral nucleus of the amygdala has also been implicated in the acquisition of cue value (8). To establish whether this aspect of associative learning is truly spared in our animals, we conducted further experiments to examine the nature of the learning acquired by cues during appetitive Pavlovian conditioning in rats with selective CN damage.

Perhaps the most frequently cited measure of a cue's changed motivational value, as a consequence of its pairing with a biologically meaningful stimulus, such as food, is its ability to reinforce subsequent learning—for example, the phenomenon known as second-order conditioning. We tested a cue's ability to support Pavlovian second-order conditioning in rats with CN damage. As shown in Fig. 1, second-order conditioning was as robust in rats with CN lesions as it was in normal control rats (see legend to Fig. 1 for description of task). Thus, CN damage did not interfere with a cue's ability to acquire reinforcing power when paired with food.

We also tested whether a cue's acquisition of motivational properties remains intact in rats with CN damage by using a procedure called "conditioned potentiation of feeding." In this procedure, sated rats are induced to eat by presenting a previously trained cue for food (9). To test for this effect, rats were placed back on ad libitum feeding after standard appetitive training and exposed repeatedly to the test chamber with food available to eliminate all tendency to consume food in that environment. Two test sessions were then conducted, during which 20 food pellets were made available. In one session the formerly trained cue was presented; in the other no cue presentations occurred during the test. Our experiment showed that feeding was potentiated substantially by the cue in normal rats; whereas an average of 0.2 pellet was consumed in the unsignaled test, on average, 15.2 pellets were consumed in the cue-signaled condition. The rats with CN damage showed a comparable effect of cue presentations; lesioned rats ate 3.8 pellets in the unsignaled condition and 16.9 pellets in the signaled condition (10).

These results provide strong support for the conclusion that the motivational value associated with cues during appetitive learning is acquired normally by rats with amygdala CN damage. Taken together with research conducted in other laboratories (e.g., refs. 6 and 8), it appears that a separate subsystem within the amygdala is necessary for this function. Despite the normal appearance of rats with CN damage on the tests described above, the lesions were highly effective in producing other behavioral impairments. One that can be observed during simple appetitive conditioning will be described here; others will be introduced in a later section of the paper.

We observed that neurotoxic damage to the amygdala CN produces a profound deficit in the acquisition of learned orienting behavior to either visual or auditory cues (7). Fig. 2 illustrates food-

Abbreviations: CN, central nucleus of the amygdala; US, unconditioned stimulus; CS, conditioned stimulus.

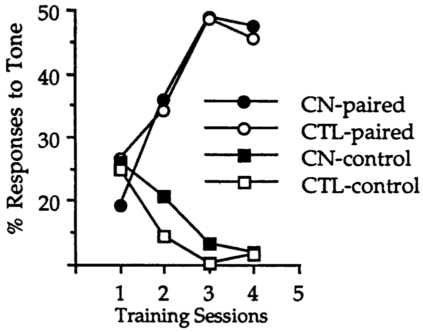

FIG. 1. Second-order Pavlovian conditioning. Rats were initially trained to approach a food cup in response to a light paired with food (first-order conditioning, data not shown). Then rats received training sessions in which a tone was either paired with the light (paired groups) or presented alone (control groups). The results for the second-order conditioning sessions in the graph show learning to the tone when it was paired with light but not when it was presented alone. This learning was equivalent in CN-lesioned rats (CN-paired) and control rats (CTL-paired).

reinforced orienting to visual cues, which consists of rearing on the hind legs and orientation toward the light source. Although the topography of learned orienting resembles the spontaneous orienting of rats to novel stimuli, CN damage did not alter spontaneous orienting. Habituation of spontaneous orienting to repeated nonreinforced presentations of those stimuli, either within single sessions or across sessions over days, was also entirely normal (7). These findings resemble those from earlier studies examining an autonomic component of the orienting response and conditioning of that response in rabbits. Damage to the CN or neuropharmacological treatments within the CN were found to prevent the acquisition of conditioned heart-rate responses while entirely sparing the occurrence of heart-rate orienting responses to novel stimuli (11–13). Thus, it appears that CN is critically involved only in the development of learned orienting responses. Because the Pavlovian task we use to study rats permits this learning to be monitored in the same experiment in which learning to approach the food-cup also occurs, we confirmed that deficits in the acquisition of orienting were evident in the previously summarized studies in which CN damage did not affect learning the motivational/reinforcing value of cues (see Fig. 2). This impairment of orienting during learning, without any change in the cue's ability to acquire reinforcing properties, led us to consider other contributions of the CN to associative processes. A description of the subsequent behavioral research we conducted will follow a section in which we consider the anatomical connectivity of the CN as a framework for understanding the function of this subsystem of the amygdala.

A Neuroanatomical Framework for CN Function

Among the nuclei of the amygdala complex, the CN is distinguished by its projections to the lower brainstem (14–16). Much research has shown that many brainstem targets of CN exert control over autonomic and behavioral responses used in the expression of associative learning. Conditioned-freezing, heart-rate, potentiation of startle and eyeblink reflexes all appear to depend on output from brainstem systems that are innervated by CN (17–20). For example, the acoustic startle-reflex circuit is organized at the level of the brainstem and includes the nucleus pontis caudalis, which receives a substantial projection from CN that is critical for potentiation of the reflex based on learning (21).

In addition to the somatomotor and autonomic effector targets of CN in the brainstem, a collection of ascending systems receive input from the CN, including monoamine systems (norepinephrine, serotonin, and dopamine), the pontomesencephalo–tegmental areas where brainstem cholinergic neurons are located, and the basal forebrain system that innervates the cortex (15, 21–25). Collectively, these ascending systems have been assigned roles in arousal, vigilance, and attentional functions. A subset of these projections, which will be discussed further, is shown in Fig. 3. We think that the CN uses these pathways to exert control over forebrain processing during learning. For example, several pathways provide potential routes for the control of learned orienting. The CN has an indirect influence on the striatum through its input to midbrain dopamine neurons in the substantia nigra (shown in Fig. 3). Considerable research indicates that dorsolateral striatum plays an important role in the initiation of responses guided by sensory events, including the regulation of orienting behavior to cues in many different modalities (e.g., refs. 26–29). It is notable that a prominent component of the projection from CN innervates a dorsal tier of dopamine neurons in the lateral substantia nigra pars compacta (30), neurons that project primarily to dorsolateral striatum (31). In addition to this circuitry, input from CN to pontomesencephalo–tegmental regions in the brainstem (LDT/PPT shown in Fig. 3) may also be involved in orienting behavior. The cholinergic neurons in these regions innervate subcortical structures that influence cortical processing (via anterior, reticular, mediodorsal, central medial, and posterior nuclear regions of thalamus) and other areas that control sensorimotor function (e.g., superior colliculus, extrapyramidal structures, etc.) (32, 33). The superior colliculus, in particular, plays an important role in visual orienting (e.g., ref. 34).

Recent neuroanatomical studies have shown that CN output also targets systems located in the basal forebrain, which in turn regulate cortical processing. This anatomy is interesting in light of recent studies indicating that the basal forebrain is involved in the allocation of attentional resources, even when overt orienting behavior is absent (35–37). A component of this basal forebrain system uses acetylcholine as a neurotransmitter. In the

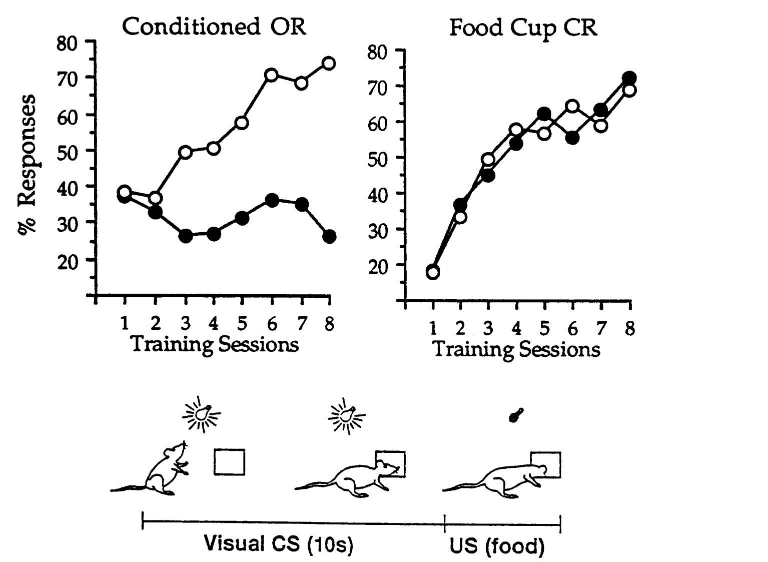

FIG. 2. (*Lower*) The cartoon illustrates the occurrence of orienting behavior (Conditioned OR) early in the CS interval and the occurrence of the learned food cup response (Food Cup CR) toward the end of the CS presentation. (*Upper*) Graphs show the performance of intact rats (○) and rats with CN lesions (●) on the learned orienting response (*Left*) and food cup response (*Right*) when a visual CS was paired with food (eight trials per session). Damage to CN only impaired learning of orienting to the CS.

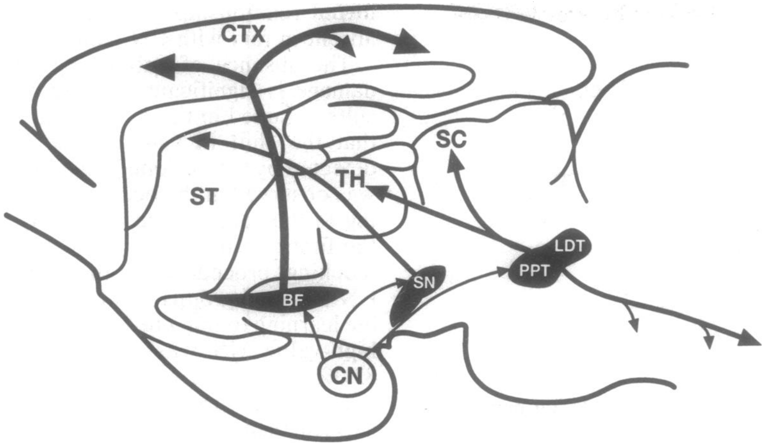

FIG. 3. Schematic of the rat brain in a parasaggital view. The CN is in the temporal lobe and projects to several systems that innervate the forebrain. The projection to the substantia nigra (SN) provides access to dopamine neurons that innervate the dorsolateral striatum (ST). Projections from CN to the lateral dorsal tegmental nucleus (LDT) and pedunculopontine nucleus (PPT) provide a route for regulating pathways to midline thalamic nuclei (TH) and superior colliculus (SC), as well as other extrapyramidal structures (data not shown). A system of magnocellular neurons in the basal forebrain (BF), which projects to the cortex (CTX), is also under the influence of CN efferents.

monkey these cholinergic neurons are concentrated in the nucleus basalis of Meynert, and efferents from CN onto cholinergic neurons in the nucleus basalis of Meynert have been identified in the primate brain (38). A homologous system of cholinergic neurons—scattered throughout the sublenticular substantia innominata and ventral globus pallidus, as well as the nucleus basalis of Meynert—also receive input from the CN in the rat (ref. 23 and illustrated in Fig. 3).

The Regulation of Attention by the CN

Progress in research on the neurobiology of attention in the past decade has provided a number of key insights. (*i*) Attention is mediated not by a single system but by a set of systems, each of which specializes in components of attention that can be specified in cognitive terms. For example, Posner and colleagues (39, 40) have defined a number of specialized functions, such as maintaining vigilance or an alert state, orienting to sensory events, and detecting signals for focal processing, which rely on distinct neural circuitry. In addition, different operations within these networks are responsible for the dynamic control of attention—i.e., disengaging attention, shifting attention, and engaging a selective focus of attention. (*ii*) Considerable evidence now indicates that these attention systems are anatomically separate from information-processing systems that deal with specific inputs from the external environment.

The neuroanatomical connectivity between the amygdala CN and other brain systems noted in the preceding section provides potential routes for regulating component processes of attention, including alerting/vigilance (through the lateral dorsal tegmental nucleus/thalamic/cortical system and ascending noradrenergic pathway), the selection of orienting responses to cues (via pedunculopontine nucleus/superior colliculus and dopamine/dorsostriatal systems), and shifts in the focus of attention (through the basal forebrain projection to cortex). The following section will deal with studies of CN function in attention. The results of these studies suggest that CN output may be particularly critical for engaging or incrementing attention to cues when expectations established by associative learning are modified.

The original impetus for our studies on the role of the CN in attention during associative learning stemmed from the observation that CN-lesioned rats failed to acquire orienting responses. Research based on modern theories of learning has indicated that conditioning episodes produce important changes in the processing of cues, as well as in the formation of associations between the conditioned stimulus (CS) and unconditioned stimulus (US) (e.g., refs. 41–44). Such changes in attention may be characterized as either incremental or decremental. For example, Pearce and Hall (42) suggested that losses of attentional processing can occur when a cue provides no new information about subsequent events, but increases in attention occur when important events happen unpredictably or when expectations about the occurrence of events are violated. By this view, the failure to learn orienting behavior might reflect a failure to increase attention to a cue when its relation to another significant event (the US) is initially detected. In subsequent studies of rats with CN damage, we found a general absence of the tendency to increase attention—for example, when contingencies between a cue and other events were altered. At the same time, no effect of CN damage was found in a variety of situations in which decremental changes in attention to cues are observed—for example, when a cue consistently signals the absence of food.

An associative learning task initially developed by Wilson *et al.* (45) was used to investigate the role of the CN in modifying attention to a cue (46). As shown in Table 1, two training conditions are used. In one condition, a "consistent" predictive relationship between a light followed by a tone occurs throughout phases 1 and 2 of training, and these trials are themselves reinforced with the food US on half the trials. In the "inconsistent" training condition, trials identical to those in the consistent procedure occur in phase 1, so that rats come to expect that the light is invariably followed by the tone, but a manipulation of the reliable relationship between these cues occurs in phase 2; the tone is unexpectedly omitted on nonreinforced trials. In phase 3 allocation of attention to the light is assessed by monitoring how readily rats learn to approach the food cup during the light when it is paired directly with food.

Wilson *et al.* (45) originally showed that the inconsistent training procedure increases attention to the light in phase 2, as evidenced by facilitated acquisition of learning in the final test phase. Fig. 4 *Left* shows this result for groups of intact rats in our study (46). Fig. 4 *Right* shows the effect of neurotoxic CN lesions. A comparison of performance across Fig. 4 *Left* and *Right* indicates comparable learning for CN-lesioned and control rats when a consistent relationship between the cues is maintained. However, instead of improved learning, the inconsistent training condition makes learning worse in rats with CN damage. It is clear that an unexpected shift in the light's predictive

Table 1. Procedures used to increment attention by altering the predictable relation between two cues

Training condition	Phase 1 (consistent light–tone relationship)	Phase 2 (experimental change in light–tone relationship)	Phase 3 (test of learning to light)
Consistent	L → T → food; L → T	L → T → food; L → T	L → food
Inconsistent	L → T → food; L → T	L → T → food; L	L → food

Half of the trials in phases 1 and 2 are reinforced with food; the other half are nonreinforced trials. In phase 3 the trials are always reinforced with food. L, light; T, tone.

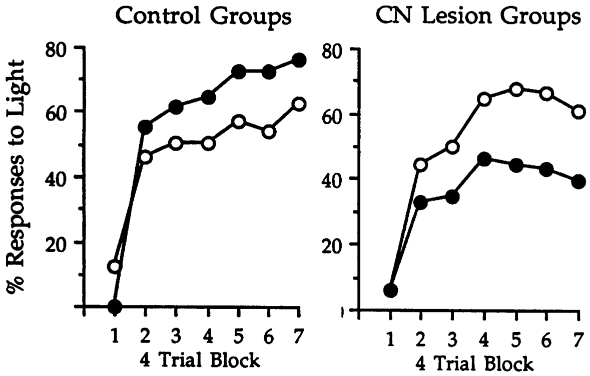

FIG. 4. Augmentation of attention by manipulating the predictive relation between two cues. Training procedures are described in Table 1. The graphs show the results from the final phase in which simple appetitive learning to the light was assessed. (*Left*) Data for intact groups (Control). (*Right*) Data for groups with CN lesions. ○, Consistent prior experience; ●, prior experience in which the relation between cues was shifted to an unpredictable condition in phase 2 (inconsistent prior experience). Note that rats normally learn better when they have experienced a change in what they expected (inconsistent vs. consistent groups). This is not the case for rats with CN damage. [Reproduced with permission from ref. 46 (copyright American Psychological Association).]

validity in phase 2 did not increase attention to the light in rats with CN damage. In the absence of increased attention to this cue, there are several reasons why rats in the inconsistent procedure would show less conditioning—e.g., less protection from extinction, habituation, or latent inhibition, all of which remain unaffected by CN lesions (refs. 7, 46, and 47; see ref. 47 for a full discussion).

Another task designed to demonstrate that altering the predictive relationship between cues can increase attentional processing yielded additional evidence that rats with CN damage are impaired in this aspect of attention. The "blocking" paradigm has been commonly used for the examination of attention in associative learning. Here, learning to a cue, such as a tone, that is presented at the same time as another cue, such as a light, is prevented or greatly diminished by prior conditioning to the light alone. Attention-based theories account for this blocking of learning in terms of how prior learning to one cue may change attention allocated to a cue that is added later in training. For example, because the significant event (the food US) is already well predicted when the compound light/tone is introduced, any attention paid to the added cue will rapidly diminish; it provides no new information. Learning the relationship between the added cue and the occurrence of the food US is blocked, therefore, by decrements in attention. Support for this account has come from the effects on learning produced by variations in the usual blocking procedure. One such variant is to change the predicted relationship between the original cue and the food US when compound conditioning is introduced. This change can be made by either increasing or decreasing the magnitude or value of the US. Both procedures augment conditioning to the newly introduced cue, a phenomenon referred to as "unblocking." In particular, the observation that greater learning occurs when the value of the US is decreased in an unblocking experiment has provided evidence that increased attention to a cue occurs when a previously established relationship between events is altered.

Unlike normal rats, we observed that rats with CN damage fail to show greater learning to a cue when unblocking is produced by decreasing the value of the US (47). Two sets of rats (one with CN lesions and a control group) were trained in each of the conditions shown in Table 2. When no training occurred before training with the compound in phase 2 (control condition), robust learning occurred to the target cue; ≈60% responses were observed in both the lesioned and control rats during the final test. By comparison, learning was blocked when the same US (either high or low value) was used throughout training in phase 1 and 2 (blocking conditions); the test for conditioning to the target cue revealed <25% responses, irrespective of lesion condition (shown in Fig. 5). Relative to the small amount of learning for these groups, a decrease in US value introduced in the phase 2 unblocking procedure improved learning to the added cue, but only in the control rats (also shown in Fig. 5). Thus, the increase in attention usually produced by changing the predicted relationship between events was absent in rats with CN damage.

The absence of unblocking after CN damage is significant because other results obtained in the experiment showed that the shift in value of the US was detected by CN-lesioned rats. This result was observed in the occurrence of learning to the original cue (the light) across the first two training phases. In the unblocking procedure, both control and CN-lesioned groups exhibited comparable learning to that cue in phase 1 when a high-value US was used (79.2% and 83.3% responses for control and CN-lesioned groups, respectively), and learned responding decreased comparably in phase 2 when the US was decreased (68.8% and 65.3% responses for control and CN-lesioned rats, respectively). Thus both lesioned and control rats were equally sensitive to changes in the US across training, but only the control rats showed unblocking. These results have been replicated in two separate experiments (47), indicating that altering the cue-reinforcement relation, while detected by rats with CN damage, does not produce an increase in attentional processing.

In contrast to the effects of CN lesions in situations where normal animals increase attentional processing of cues, such damage does not affect decremental attentional processes. This result is evident in habituation of spontaneous orienting to neutral cues, in blocking itself, and in the effects of repeated exposure to cues on subsequent conditioning (e.g., latent inhibition), all of which proceed normally in rats with neurotoxic lesions of the CN (7, 44). Interestingly, effects on those functions spared by CN damage are often impaired by lesions of another brain system studied in associative learning, the hippocampal formation including the septohippocampal pathway (48–52). This result raises the possibility that incremental and decremental regulation of attention is served by separate neural systems. We turn now to a preliminary exploration of how the regulation of attention by CN may be mediated through its connectivity with systems identified in the previous section of this review.

The concept that the pathway from CN to the basal forebrain (shown in Fig. 3) provides a substrate for the regulation of attention is consistent with other evidence for a role of the basal forebrain in attentional processes cited earlier (35–

Table 2. Unblocking experiment

Group	Training procedure	Phase 1	Phase 2	Test
Control	High US, phase 2 only		Light/tone → US_H	Tone
Blocking	High US, phases 1 and 2	Light → US_H	Light/tone → US_H	Tone
Blocking	Low US, phases 1 and 2	Light → US_L	Light/tone → US_L	Tone
Unblocking	High US shifted to low US	Light → US_H	Light/tone → US_L	Tone

US_H, high-value US; US_L, low-value US; →, cued paired with US.

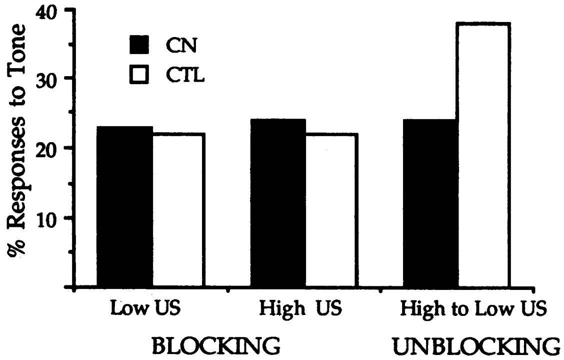

FIG. 5. Unblocking produced by decreasing the value of the US when training with the compound cue was introduced. Training conditions conform to the procedures shown in Table 2. The intact rats (CTL group) in the unblocking condition exhibit more learning than rats in the blocking conditions. This effect is not seen in rats with CN lesions. [Reproduced with permission from ref. 47 (copyright American Psychological Association)].

37). In addition, amygdala CN regulation of the projection from basal forebrain to cortex has been recently studied. Whalen *et al.* (53) found that classically conditioned neocortical activation of the electroencephalogram (which is sensitive to drugs that block cholinergic function) is tightly coupled to conditioned neural activity in the basal forebrain. The possibility that this associative regulation of basal forebrain activity is controlled by the CN is supported by evidence that CN stimulation elicits the cortical activation mediated by cholinergic input, and neurotoxic CN lesions abolish learned activation of the cortical electroencephalogram (54, 55).

In a recent experiment, we examined the effect of basal forebrain lesions, induced by microinfusion of the neurotoxin α-amino-3-hydroxy-5-methylisoxalzole-4-propionic acid (AMPA) into this region (56). These rats along with appropriate control groups were tested in the task devised by Wilson *et al.* (45). We found that this damage reproduced the effect of CN lesions; rats with basal forebrain damage failed to increase attention when the relationship between events was unexpectedly modified. More recently we have removed only the neurons in the basal forebrain that provide cholinergic innervation of the cortex using a newly developed immunotoxin, 192 IgG-saporin (57). These selective lesions also reproduced the deficit in attentional regulation seen after CN damage (A. Chiba, M.G., and P.C.H., unpublished work). One result found with CN damage, however, was not evident in rats with basal forebrain lesions—namely, there was no significant effect of this damage on the occurrence of learned orienting behavior. Thus, increased attentional processing of target cues may involve a CN–basal forebrain pathway, but mediation of learned orienting must use different circuitry. As suggested earlier, that orienting behavior might depend on the CN/nigral/dorsolateral striatal system. Rats with damage to the dorsolateral striatum or its dopamine innervation fail to orient spontaneously to cues (27–29). Although CN damage, unlike striatal damage, does not affect spontaneous orienting to novel cues, CN output might engage this system for learned orienting behavior.

Conclusion

The suggestion that the amygdala complex is involved in the regulation of attention is not entirely novel (see ref. 58). Until recently this concept has rested on only a few fragmentary observations. Earlier research described aimless visual tracking in monkeys with amygdala damage (59), and an alerting/orienting reaction evoked by electrical stimulation at sites in the vicinity of amygdala CN was originally described in cats (60). More recently, the notion that amygdala CN regulates arousal during learning has been proposed (61). The neuropsychological perspective of CN offered here may help to broaden our view of amygdala function, within a more clearly defined neuroanatomical and behavioral framework for the study of attention. In addition to the amygdala's long-acknowledged role in associative learning of emotional responses, it is suggested that the CN regulates the processing of cues when predictive relationships between events are first noticed or altered. In particular, the allocation of attention normally engaged when an expectation about the occurrence of events is violated does not happen after damage to this system. This role of the CN has potential implications for a number of clinical situations involving attentional disorders where the biological basis is poorly defined, as well as for the neuropsychological study of patients with known amygdala damage.

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